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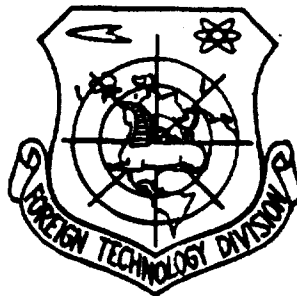


AEROGENIC DISSEMINATION
OF APHTAE EPIZOOTICAE

by

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AEROGENIC DISSEMINATION OF APHTAE EPIZOOTICAE

Jerzy Wisniewski

During aphtae epizooticae the principal cause of the virus dissemination is animate and inanimate factors. These factors are contacts between sick and susceptible animals, mechanical dissemination by man, animals, birds, insects, aerial dissemination in cattle purchasing centers, transport, and contaminated products such as milk and butter.

Besides the above factors of the aphtae epizooticae viruses, there is also the factor of virus dissemination in aerosol elementary particles. This aspect has neither been studied nor mentioned in the Polish scientific literature [26, 27, 28, 29, 34, 48, 49].

Until the catastrophic aphtae epizooticae in Great Britain in 1967-1968, only a few authors reported cases of aphtae epizooticae dissemination as a result of air movement caused by meteorologic phenomena [16, 22, 31, 35, 36, 50]. As a result of huge economic losses caused by the above mentioned epizootic, which spreads with unusual intensity, British virologists undertook complex studies in order to determine the reason for such an unexpected spread of aphtae epizooticae. The main role in these studies belongs to the Pirbright Institute [4, 5, 6, 10, 11, 12, 13, ^{14, 17}39, 40, 41, 42, 43, 44, 45].

Several decades ago it was proven that animal illness emits enormous amounts of aphtose virus into the environment, whose main source is bladder epithelium along with lymphy, saliva, milk, urine, excrement, and seeds. The above excretions contain the virus at least several hours before

blisters show up. The virus achieves maximum concentration during generalized morbid symptoms.

Last decade; owing to the application of special equipment [30] enabling collection of large capacity air samples, and collection of the virus in buffer gas; proved that the animals suffering aphtae epizooticae, such as cattle, sheep, and pigs, exhale significant amounts of virus [14, 22, 45]. Similar to the excretions, the virus appears in exhaled air several hours after infection and its maximum concentration develops just before the boils break. Cattle and sheep emit $3 \times 10^4 \text{ID}_{50}$ of the aphtosa virus, whereas pigs emit 10^6ID_{50} which is about 30 times higher than that of cattle. The studies conducted to determine the presence of the virus in the air samples taken from boxes where sick pigs were kept, produced $10^{7.2} \text{ID}_{50}$ of the virus [44]. A liter of the air exhaled by a sick calf contained 6.3 to 630 ID_{50} of the aphtosa virus [24]. ~~The~~ period of virus emitted along with exhaled air lasts from several hours to a dozen days. ^{22, 45}

What is the origin of the virus which settles in the air? According to Korn's opinion the proliferation of the aphtosa virus takes place mainly in the mucous membranes of the upper respiratory tract, namely in the nasal mucus, throat mucus, Highmore antrum, trachea and even in bronchus [25]. Eskildzen states that the aphtosa virus can also proliferate in the epithelium of lung blisters at the incubation stage [15]. Shortly after infection, the aphtosa virus is found in the excretion of the mucous membrane of the upper respiratory tract as well as in the flow of exhaled air. Aerosols carrying the virus can be found when fodder is chewed, when smacking, coughing, and mooing. The saliva foam assists in spreading the virus in the air. The aerosol containing the

virus can also be found in evaporation from wet floors, as well as in infected excrement and urine [7], in stable manure or even when the manure is scattered by a sprinkling machine [8, 39]. In Great Britain one assumes that aerosols carryin the virus, during the 1967-1968 epizootic arose as a result of delivering milk from infected farms when milk was mechanically pumped from milk-cans to tank-cars [8].

The virus can remain in the air due to the microscopic size of aerosol particles. May proved that 70% of the virus found in the samples of air studied was connected with aerosol particles exceeding 6 microns, 20% was connected with particles of 3 to 6 microns; 10% was connected with particles of less than 3 microns. The aerosol, which is carried by air, can be removed by settling to the ground, or by wasing by rain, snow, or fog [9, 17, 19, 47]. When the weather is rainy and wet the speed of particle settlement is 10 times faster than when the weather is dry [47]. On windless days the aerosol particles carrying the virus settle to the ground due to gravity. The size of aerosol particles determines the time it takes for dissemination. The optimum conditions for virus dissemination are represented by aerosols whose particle size is 2 to 10 microns. When conditions are favorable, (the air is very humid and the wind blows at night), the virus may be disseminated at distances of even several dozen kilometers [3, 7, 21, 31, 33]. Knowing the speed of the wind, the number of sick animals, one can calculate (on the basis of the Pasquille formula) the virus concentration in the air at different distances from the center of the epidemic (according to 45).

The aphtosa virus can penetrate into a susceptible animal through the alimentary canal or respiratory tract. The areas for virus penetration are mucous membranes and, rarely,

skin. Until recently we have assumed that the aphtosa virus induces infection through the mucous of the alimentary canal. Studies involving the susceptibility of the infection areas proved that an oral infectious dose for cattle amounts to 10^6ID_{50} , for pigs it goes up to 10^5ID_{50} of the virus. A nasal or endotracheal infectious dose for cattle amounts to

Table 1. The maximum amount of virus collected from an animal; time and range of pathogenic changes in the animals infected by strains of the aphtosa virus (Donaldson, Herniman, Parker, Sellers, 1970 [14])

Virus strain	cattle		sheep		pigs		total amount of virus collected
	maximum amount of virus	time & range of pathogenic changes (hours)	maximum amount of virus	time & range of pathogenic changes (hours)	maximum amount of virus	time & range of pathogenic changes (hours)	
O ₁	3.7 *)	41, G	3.6	24, N	5.4	45, G	12.7
O ₂	2.5	42, G	2.1	22, N	5.1	68, G	9.7
A ₁	2.8	46, G	1.7	44, P	4.7	48, G	9.2
A ₂₂	2.8	70, G	1.35	19, N	4.25	48, G	8.4
C _{LEB}	2.7	93, G	1.5	19, N	4.35	46, G	8.55
C _{NOV}	3.3	70, G	3.7	22, N	5.6	46, G	12.6

Explanations: *) total amount of the virus (log ID_{50}) collected from the animal during 60 minutes in 1000 liters per minute; N - lack of pathogenic changes; P - initial pathogenic changes; G - generalized pathogenic changes.

10 to 100 ID_{50} of the aphtosa virus [15, 22, 39, 45]. When the virus is disseminated in an aerogenic way, the cattle become infected faster than anything else because of inhalation of the maximum amount of air. Norris and Harper are of the opinion that aerogenic virus dissemination can infect either through the respiratory tract or the alimentary channel [33]. Virus carrying aerosols, accumulated in cow-sheds where the animals suffering with aphtae epizooticae are kept, can get outside. This may be the result of a draft caused by an open window or door, however, it is mostly likely caused by ventilation systems. The process of air escape through ventilators can be seen in the winter when the outside

temperature is low, when the warm air from cow-sheds evaporates in the form of a mist strip carried by the wind. If a significant amount of the virus carrying aerosol gets outside and if there are favorable conditions for the virus to survive, then it can be carried away at significant distances by the wind current and cause infection in another place.

Virus survival in the air depends on several factors, namely - on the virus strain, on the chemical composition of the virus suspension, on the air temperature, sun activity, and especially on relative humidity [4, 5, 6, 11, 12, 13]. The virus strains originating from areas with dry climates such as Pacheco (Argentina) designated as 0-1, Irak as A-22, Lebanon as C have more stable aerosols when compared to moderate climates such as Lombardia as 0-1, Brescia as 0-2, England as 0-1 BFS [10]. The virus obtained from milk, excrement suspension, or from cell culture fluid is more stable than the one obtained from virus carrying aerosols originating in saliva [11]. The open air factor as well as sunlight during fall and winter causes insignificant degrees of virus inactivation [12]. This property is typical for the entire group of the picornaviruses [46].

The survival of the aphtosa virus in aerosols depends mostly on relative humidity [4, 5, 11, 12, 40]. Different strains of the aphtosa virus being held at 20°C with relative humidity above 60%, reveal the maximum infecting strength. After 5 minutes in 60% relative humidity the aerosol retains 12% of the initial infecting strength of the virus, further slow inactivation process takes place after 24 hours. The degree of inactivation after an hour hold of virus-carrying aerosols in 70% relative humidity of the air differs significantly depending on the virus strain and can be within the

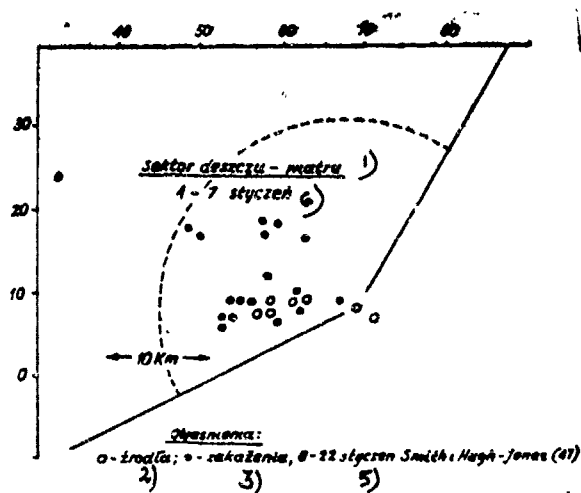


Fig. 1. Scheme of the Hampshire epizootic (1967)

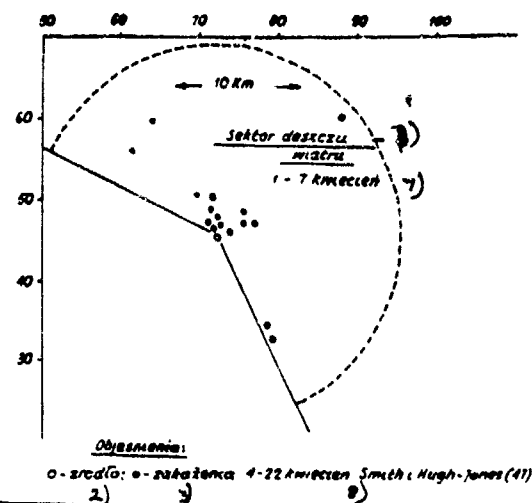


Fig. 2. Scheme of the Cheshire epizootic (1952)

Key for both figures: 1) rain and wind sector, 2) source, 3) infections, 5) January 8-22, Smith and Hugh Jones [47]. 6) January 4-7, 7) April 1 to 7; 8) April 4 to 22, Smith, Hugh Jones [47].

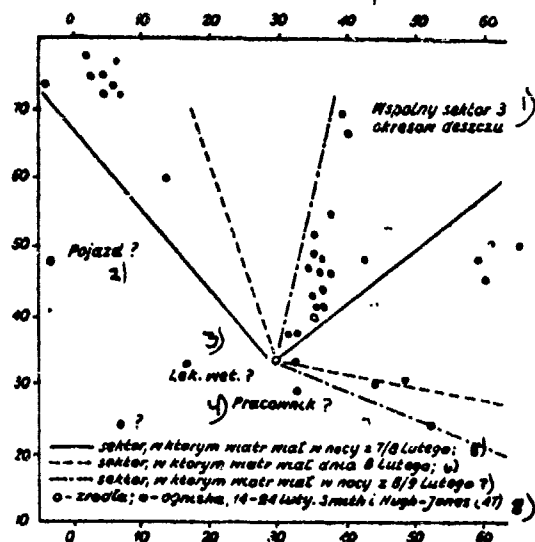
1.1 to 3.2 log range [10]. The virus drying in a condition of low relative humidity (below 20%) results in faster disactivation [10, 11]. When the relative humidity exceeds 70% and there is a lack of sunlight, one can expect that the virus can survive in the air at least for several hours, and sometimes more than ten hours [12, 39, 45, 51].

Observation of different epizootic centers of aphtosa, and even significant epizootics indicates that the virus aerogenic transmission when the meteorologic conditions are favorable, presents a dangerous method of infection. In the world scientific literature of the last decade there have been many publications describing and analyzing the reasons and course of the determined epizootics as well as the effect of meteorologic conditions on the spread of aphtosa virus [3, 17, 19, 43, 47]. Some of these deal with the reasons of

sudden spread of aphtosa during the epizootic of the 0-1 type virus in Great Britain in the fall of 1967. Brooksby's opinion assumes that the reason for the aerogenic spread of the virus was the increase of the epizootic centers of aphtosa with reference to the wind direction [8]. Wind-blown farms were located on the map in the area outlined by the V whose arms merged in the center of the primary epizootic.

On the basis of the general analysis of the epizootic centers' localization during the epizootic in Worcestershire, Henderson showed the aphtosa spread along the wind-blown side within the 3 mile area [17]. The closer to the epizootic center, that is, in the area of stronger concentration of virus in the air, the higher the percentage of recurrence and the more uniformly aphtosa is spread. The further from the epizootic center the area is, the smaller the number of infected farms. The cases where the disease was disseminated by human beings, birds, and vehicles were only sporadic.

In connection with the suspicion of aerogenic spread of aphtosa epizootic, scientists from the Central Laboratory of Veterinary Science in Weybridge along with meteorologists and physicists from Harwel studied retrospectively the aphtosa spread in Oswestry in 1961, in Cheshire in 1952, in Northumberland in 1966, and Hampshire in 1967. During the above epizootics there were 280 aphtosa centers recorded, from these, only 15 centers were within the area of rain and wind (Fig. 1, 2, 3). Analysis of the epizootics indicated that the direction of wind and rain had turned out to be a decisive factor ^(90% in) of the localization of the disease centers [47]. Discussing the reasons for the epizootic of aphtosa in Great Britain in 1967-1968, Hyslop assumes that the principal reason for the failure to fight the disease was enormous virulence of the 0-1 type, the sudden spread of



Key: 1) joint sector of 3 periods of rain, 2) vehicle?, 3) veterinary surgeon?, 4) worker?, 5) sector where the wind blew at night, Feb. 7-8, 6) sector where the wind blew during the day, Feb. 8, 7) sector where the wind blew at night, Feb. 8-9, 8) a - sources, b - centers, February 14-24, Smith and Hugh-Jones [47].

Fig. 3. Scheme of the Owestry epizootic (1961). Recurrent stage: wind and spread sector.

aphtosa in pig-farms strong winds blowing in the direction of areas with mass farming of susceptible animals as well as cool rainy weather. All these reasons resulted in retention of the infectiousness of the mobile virus [21].

Other European countries were also plagued by the aerogenic transmission of the virus. The winter of 1965-1966 became the winter of the epizootic which significantly reduced the Swiss livestock population. In this winter the wave of aphtosa spread in the form of succeeding jumps from Switzerland's South-West towards its North. The aphtosa spread was proportional to wind intensity. Aphtosa spread despite very strict rigors established from the very beginning of the epizootic.

Clinical signs in cattle showed up after 2 to 3 days of windy weather. The way aphtosa spread was the best proof that the speed, direction and localizations of the epizootic centers depended on the strength of the wind but not on

regional travel. Analysis of the cases of aphtosa performed crosswise valleys which did not run across the land confirmed the above-mentioned. Aphtosa persisted mostly on the tops and gradually decreased on the slopes towards valley bottoms. The direct transmission of aphtosa from one top to another was faster than the aphtosa crosswise transmission through a valley bottom [7, 37].

The aerogenic transmission of aphtosa virus can be slowed by a dense cloud cover. A similar case was described by Bischofberger. It took place in the mountainous region of Appenzell [7]. During the epizootic there were no aphtae epizoticae centers in this area above the 1,000 m level, and at the same time not too far away from this region aphtosa persisted among many animals. The analysis of the influence of atmospheric conditions on the range of the aphtosa centers, which was done with the cooperation of nearby meteorologic stations, indicated that during the aphtosa spread dense clouds and fog prevailed in the area reaching the 1000 meter ceiling. When the cloud layer went up to the 1,100 meter level, the aphtosa center appeared on this level 2 days after it happened. Bischofberger assumes that the aerogenic dissemination of the aphtosa virus takes place when the virus infectiousness exceeds the normal level, when there is a mass source of the virus and certain defined meteorological conditions came into being. A similar group of factors corresponded to the epizootics in Switzerland in the years of 1920-1921, 1938-1939, and 1965-1966 [7].

In GDR one observed the avalanche recurrence of aphtosa centers spreading according to the wind direction [50]. It was assumed that the reason for the sudden explosion of the number of these centers lay on the virus precipitated on dust and transmitted by wind current. The present science with its knowledge explains that aerogenic transmission of

aphtose is due to virus travel on aerosol microparticles. The adsorption of the virus on hard particles, such as dust, pollen or bacteria does not necessarily represent the reason for aerogenic transmission of the virus. These particles are one of the factors of aphtosa dissemination [49].

The phenomenon of virus transmission by aerogenics has been observed many times in Sweden, Norway, and Denmark. The aphtosa virus was transmitted by wind blowing from West Germany and GDR in a northerly direction [16, 31]. The first centers of the disease in Denmark appeared on the southern shores of the islands at a 25 to 30 mile distance from the land border with West Germany. The time period excluded the possibility of disease transmission by migrating birds.

Cases of virus transmission through the air were also observed in institutions studying the virus and producing vaccinations for aphtosa (1, 18, 21, 31). In January, 1960 just two miles from the Pirbright Institution the SAT-2 type aphtosa broke out. The detailed study indicated that the virus escaped from the Institution isolation wards where the live-stock infected by the SAT-2 type virus were kept. The virus reached the farm by air [21]. At that particular time the isolation wards were not yet equipped with air filters. The second case of aerogenic transmission of the virus in the same Institution happened in 1967 when the virus escaped because of damage to the system of air filters in the section with infected animals [21].

A similar case of escape of the 0-1 type virus from the Institution on Lindholm Island in Denmark was described by Michelsen in 1968 [31]. The virus transmitted by the air caused infection of live-stock within a 5 km range from the Institution. The infection was spread on the leeward side.

During 2 weeks there were several recurring centers in this area as well as one center in Sweden - 40 miles away from the initial center. The late fall, stormy weather, and lack of any contacts prove that the transmission of the virus was aerogenic.

In conclusion, one can prove that the aerogenic transmission of the virus is one of the most essential ways of the dissemination of aphtosa. Sick animals, no matter what virus type it is, while exhaling, release significant amounts of virus which is transmitted on aerosol microparticles. As a result of changes of the atmospheric pressure and air movement, the virus-carrying aerosols are transmitted for different distances, and the epizootic result of this phenomenon will depend on the action of different factors. These factors can include the amount of virus released, the degree of its infectiousness, a particular area's topography, and the period of the year, as well as meteorologic conditions connected with this particular period. All these factors are accountable for the virus's survival and the aerosol's volatility. Also, in addition to the above factors, the congestion of the susceptible live-stock increases the possibility of contact between the virus and the feeder.

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